

## AMENDMENTS

### In the specification:

Please amend paragraph [0186] on page 56 of the specification as follows:

[0186] In a preferred embodiment, the immunomodulatory agent decreases the amount of IL-9. In a more preferred embodiment, the immunomodulatory agent is an antibody (preferably a monoclonal antibody) or fragment thereof that immunospecifically binds to IL-9 (see e.g., U.S. Patent Application No. [[ ]]10/823,810 filed April 12, 2004 entitled "Methods of Preventing or Treating Respiratory Conditions" by Reed (Attorney Docket No. 10271-113-999), U.S. Patent Application No. [[ ]]10/823,253 filed April 12, 2004 entitled "Recombinant IL-9 Antibodies and Uses Thereof" by Reed (Attorney Docket No. 10271-112-999), and U.S. Patent Application No. [[ ]]11/105,268 filed April 12, 2004 entitled "Anti-IL-9 Antibody Formulations and Uses Thereof" by Reed (Attorney Docket No. 10271-126-999), all of which are incorporated by reference herein in their entireties. Although not intending to be bound by a particular mechanism of action, the use of anti-IL-9 antibodies neutralizes IL-9's biological effect and, thereby, blocks or decreases inflammatory cell recruitment, epithelial or neointimal hyperplasia, and mucin production of epithelial cells.

### In the abstract:

Please amend the abstract as follows:

The present invention relates to methods and compositions designed for the treatment, management, or prevention of a non-neoplastic hyperproliferative cell or excessive cell accumulation disorders, particularly those involving hyperproliferation of epithelial or endothelial cells. ~~In one embodiment, the methods of the invention comprise the administration of an effective amount of one or more EphA2 agonistic agents that bind to EphA2 and increase EphA2 cytoplasmic tail phosphorylation and/or increase EphA2 autophosphorylation in cells which EphA2 has been agonized. In another embodiment, the methods of the invention comprise the administration of an effective amount of one or more EphA2 agonistic agents that bind to EphA2 and reduce EphA2 activity (other than autophosphorylation). In another embodiment, the~~

methods of the invention comprise administration of an effective amount of one or more EphA2-agonistic agents that bind to EphA2 and decrease a pathology-causing cell phenotype (e.g., a pathology-causing epithelial cell phenotype or a pathology-causing endothelial cell phenotype). In another embodiment, the methods of the invention comprise the administration of an effective amount of one or more EphA2-agonistic agents that are EphA2-antibodies that bind to EphA2 with a very low  $K_{off}$  rate. In preferred embodiments, agents of the invention are monoclonal antibodies. The invention also provides pharmaceutical compositions comprising one or more EphA2-agonistic agents of the invention either alone or in combination with one or more other agents useful in therapy for non-neoplastic hyperproliferative cell or excessive cell accumulation disorders.